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NEWS 7	May 07	DGENE Reload
NEWS 8	Jun 20	Published patent applications (A1) are now in USPATFULL
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NEWS 10	Aug 23	In process records and more frequent updates now in MEDLINE
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FILE 'HOME' ENTERED AT 13:26:57 ON 15 SEP 2001

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=> s assay method

L1 6001 ASSAY METHOD

=> s XRCC4

L2 141 XRCC4

=> s DNA ligase IV

L3 122 DNA LIGASE IV

=> s l1 and (binding)

L4 636 L1 AND (BINDING)

=> s l2 and l3

L5 78 L2 AND L3

=> s DNA-PKcs

L6 391 DNA-PKCS

=> s l5 and l6

L7 28 L5 AND L6

=> d his

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FILE 'MEDLINE, BIOSIS' ENTERED AT 13:27:12 ON 15 SEP 2001
L1 6001 S ASSAY METHOD
L2 141 S XRCC4
L3 122 S DNA LIGASE IV
L4 636 S L1 AND (BINDING)
L5 78 S L2 AND L3
L6 391 S DNA-PKCS
L7 28 S L5 AND L6

=> s l4 and l7

L8 0 L4 AND L7

=> d l7 ti abs ibib 1-10

L7 ANSWER 1 OF 28 MEDLINE
TI Effects of DNA nonhomologous end-joining factors on telomere length and chromosomal stability in mammalian cells.
AB DNA repair by nonhomologous end-joining (NHEJ) relies on the Ku70:Ku80 heterodimer in species ranging from yeast to man. In Saccharomyces cerevisiae and Schizosaccharomyces pombe, Ku also controls telomere functions. Here, we show that Ku70, Ku80, and **DNA-PKcs** , with which Ku interacts, associate in vivo with telomeric DNA in several human cell types, and we show that these associations are not

significantly affected by DNA-damaging agents. We also demonstrate that inactivation of Ku80 or Ku70 in the mouse yields telomeric shortening in various primary cell types at different developmental stages. By contrast, telomere length is not altered in cells impaired in **XRCC4** or **DNA ligase IV**, two other NHEJ components. We also observe higher genomic instability in Ku-deficient cells than in **XRCC4**-null cells. This suggests that chromosomal instability of Ku-deficient cells results from a combination of compromised telomere stability and defective NHEJ.

ACCESSION NUMBER: 2001471542 IN-PROCESS
DOCUMENT NUMBER: 21407744 PubMed ID: 11516951
TITLE: Effects of DNA nonhomologous end-joining factors on telomere length and chromosomal stability in mammalian cells.
AUTHOR: d'Adda di Fagagna F; Hande M P; Tong W; Roth D; Lansdorp P M; Wang Z; Jackson S P
CORPORATE SOURCE: Wellcome/CRC Institute, University of Cambridge, Tennis Court Road, CB2 1QR, Cambridge, United Kingdom.
SOURCE: CURRENT BIOLOGY, (2001 Aug 7) 11 (15) 1192-6. Journal code: B44; 9107782. ISSN: 0960-9822.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20010823
Last Updated on STN: 20010823

L7 ANSWER 2 OF 28 MEDLINE

TI Genetic evidence for the involvement of **DNA ligase IV** in the DNA-PK-dependent pathway of non-homologous end joining in mammalian cells.

AB Cells of vertebrates remove DNA double-strand breaks (DSBs) from their genome predominantly utilizing a fast, **DNA-PKcs**-dependent form of non-homologous end joining (D-NHEJ). Mutants with inactive **DNA-PKcs** remove the majority of DNA DSBs utilizing a slow, **DNA-PKcs**-independent pathway that does not utilize genes of the RAD52 epistasis group, is error-prone and can therefore be classified as a form of NHEJ (termed basic or B-NHEJ). We studied the role of **DNA ligase IV** in these pathways of NHEJ. Although biochemical studies show physical and functional interactions between the **DNA-PKcs**/Ku and the **DNA ligase IV/Xrcc4** complexes suggesting operation within the same pathway, genetic evidence to support this notion is lacking in mammalian cells. Primary human fibroblasts (180BR) with an inactivating mutation in **DNA ligase IV**, rejoined DNA DSBs predominantly with slow kinetics similar to those observed in cells deficient in **DNA-PKcs**, or in wild-type cells treated with wortmannin to inactivate DNA-PK. Treatment of 180BR cells with wortmannin had only a small effect on DNA DSB rejoining and no effect on cell radiosensitivity to killing although it sensitized control cells to 180BR levels. This is consistent with **DNA ligase IV** functioning as a component of the D-NHEJ, and demonstrates the unperturbed operation of the **DNA-PKcs**-independent pathway (B-NHEJ) at significantly reduced levels of **DNA ligase IV**. In vitro, extracts of 180BR cells supported end joining of restriction endonuclease-digested plasmid to the same degree as extracts of control cells when tested at 10 mM Mg(2+). At 0.5 mM Mg(2+), where only **DNA ligase IV** is expected to retain activity, low levels of end joining (approximately 10% of 10 mM) were seen in the control but there was no detectable activity in 180BR cells. Antibodies raised against **DNA**

ligase IV did not measurably inhibit end joining at 10 mM Mg(2+) in either cell line. Thus, in contrast to the situation in vivo, end joining in vitro is dominated by pathways with properties similar to B-NHEJ that do not display a strong dependence on **DNA ligase IV**, with D-NHEJ retaining only a limited contribution. The implications of these observations to studies of NHEJ in vivo and in vitro are discussed.

ACCESSION NUMBER: 2001265822 MEDLINE
DOCUMENT NUMBER: 21189577 PubMed ID: 11292837
TITLE: Genetic evidence for the involvement of **DNA ligase IV** in the DNA-PK-dependent pathway of non-homologous end joining in mammalian cells.
AUTHOR: Wang H; Zeng Z C; Perrault A R; Cheng X; Qin W; Iliakis G
CORPORATE SOURCE: Department of Radiation Oncology, Division of Experimental Radiation Oncology, Kimmel Cancer Center, Jefferson Medical College, Philadelphia, PA 19107, USA.
CONTRACT NUMBER: 42026 (NCI)
56706
P30-CA56036
SOURCE: NUCLEIC ACIDS RESEARCH, (2001 Apr 15) 29 (8) 1653-60.
Journal code: O8L; 0411011. ISSN: 1362-4962.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607

L7 ANSWER 3 OF 28 MEDLINE
TI Mammalian Ku86 protein prevents telomeric fusions independently of the length of TTAGGG repeats and the G-strand overhang.
AB Ku86 together with Ku70, **DNA-PKcs**, **XRCC4** and **DNA ligase IV** forms a complex involved in repairing DNA double-strand breaks (DSB) in mammals. Yeast Ku has an essential role at the telomere; in particular, Ku deficiency leads to telomere shortening, loss of telomere clustering, loss of telomeric silencing and deregulation of the telomeric G-overhang. In mammals, Ku proteins associate to telomeric repeats; however, the possible role of Ku in regulating telomere length has not yet been addressed. We have measured telomere length in different cell types from wild-type and Ku86-deficient mice. In contrast to yeast, Ku86 deficiency does not result in telomere shortening or deregulation of the G-strand overhang. Interestingly, Ku86-/- cells show telomeric fusions with long telomeres (>81 kb) at the fusion point. These results indicate that mammalian Ku86 plays a fundamental role at the telomere by preventing telomeric fusions independently of the length of TTAGGG repeats and the integrity of the G-strand overhang.

ACCESSION NUMBER: 2001215859 MEDLINE
DOCUMENT NUMBER: 21151105 PubMed ID: 11256607
TITLE: Mammalian Ku86 protein prevents telomeric fusions independently of the length of TTAGGG repeats and the G-strand overhang.
AUTHOR: Samper E; Goytisolo F A; Slijepcevic P; van Buul P P; Blasco M A
CORPORATE SOURCE: Department of Immunology and Oncology, Centro National de Biotecnologia, Spain.
SOURCE: EMBO Rep, (2000 Sep) 1 (3) 244-52.
Journal code: D0T; 100963049. ISSN: 1469-221X.

PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010425
Last Updated on STN: 20010425
Entered Medline: 20010419

L7 ANSWER 4 OF 28 MEDLINE
TI Novel functional requirements for non-homologous DNA end joining in Schizosaccharomyces pombe.
AB DNA double strand break (DSB) repair by non-homologous end joining (NHEJ) in mammalian cells requires the Ku70-Ku80 heterodimer, the DNA-PK catalytic subunit **DNA-PKcs**, as well as **DNA ligase IV** and **Xrcc4**. NHEJ of plasmid DSBs in Saccharomyces cerevisiae requires Ku, **Xrcc4** and **DNA ligase IV**, as well as Mre11, Rad50, Xrs2 and DNA damage checkpoint proteins. Saccharomyces cerevisiae Ku is also required for telomere length maintenance and transcriptional silencing. We have characterized NHEJ in Schizosaccharomyces pombe using an extrachromosomal assay and find that, as anticipated, it is Ku70 and **DNA ligase IV** dependent. Unexpectedly, we find that Rad32, Rad50 (the S.pombe homologues of Mre11 and Rad50, respectively) and checkpoint proteins are not required for NHEJ. Furthermore, although S.pombe Ku70 is required for maintenance of telomere length, it is dispensable for transcriptional silencing at telomeres and is located throughout the nucleus rather than concentrated at the telomeres. Together, these results provide insight into the mechanism of NHEJ and contrast significantly with recent studies in S.cerevisiae.
ACCESSION NUMBER: 2001202768 MEDLINE
DOCUMENT NUMBER: 21145441 PubMed ID: 11226171
TITLE: Novel functional requirements for non-homologous DNA end joining in Schizosaccharomyces pombe.
AUTHOR: Manolis K G; Nimmo E R; Hartsuiker E; Carr A M; Jeggo P A; Allshire R C
CORPORATE SOURCE: MRC Cell Mutation Unit, University of Sussex, Falmer, Sussex BN1, UK.
SOURCE: EMBO JOURNAL, (2001 Jan 15) 20 (1-2) 210-21.
Journal code: EMB; 8208664. ISSN: 0261-4189.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010417
Last Updated on STN: 20010417
Entered Medline: 20010412

L7 ANSWER 5 OF 28 MEDLINE
TI Binding of inositol phosphate to DNA-PK and stimulation of double-strand break repair.
AB In mammalian cells, double-strand breaks in DNA can be repaired by nonhomologous end-joining (NHEJ), a process dependent upon Ku70/80, **DNA-PKcs**, **XRCC4**, and **DNA ligase IV**. Starting with HeLa cell-free extracts, which promote NHEJ in a reaction dependent upon all of these proteins, we have purified a novel factor that stimulates DNA end-joining in vitro. Using a combination of phosphorus NMR, mass spectroscopy, and strong anion exchange chromatography, we identify this factor as inositol hexakisphosphate (IP6). Purified IP6 is bound by DNA-PK and specifically stimulates DNA-PK-dependent end-joining in vitro. The involvement of inositol phosphate in DNA-PK-dependent NHEJ is of particular interest since the catalytic domain of **DNA-PKcs** is similar to

that found in the phosphatidylinositol 3 (PI 3)-kinase family.
ACCESSION NUMBER: 2000478876 MEDLINE
DOCUMENT NUMBER: [redacted]33533 PubMed ID: 11030616
TITLE: Binding of inositol phosphate to DNA-PK and stimulation of double-strand break repair.
AUTHOR: Hanakahi L A; Bartlet-Jones M; Chappell C; Pappin D; West S
C
CORPORATE SOURCE: Imperial Cancer Research Fund, South Mimms, Hertfordshire, United Kingdom.
SOURCE: CELL, (2000 Sep 15) 102 (6) 721-9.
Journal code: CQ4. ISSN: 0092-8674.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001030

L7 ANSWER 6 OF 28 MEDLINE
TI Interactions of the **DNA ligase IV-XRCC4** complex with DNA ends and the DNA-dependent protein kinase.
AB The DNA-dependent protein kinase (DNA-PK), consisting of Ku and the DNA-PK catalytic subunit (**DNA-PKcs**), and the **DNA ligase IV-XRCC4** complex function together in the repair of DNA double-strand breaks by non-homologous end joining. These protein complexes are also required for the completion of V(D)J recombination events in immune cells. Here we demonstrate that the **DNA ligase IV-XRCC4** complex binds specifically to the ends of duplex DNA molecules and can act as a bridging factor, linking together duplex DNA molecules with complementary but non-ligatable ends. Although the DNA end-binding protein Ku inhibited DNA joining by **DNA ligase IV-XRCC4**, it did not prevent this complex from binding to DNA. Instead, **DNA ligase IV-XRCC4** and Ku bound simultaneously to the ends of duplex DNA molecules. **DNA ligase IV-XRCC4** and **DNA-PKcs** also formed complexes at the ends of DNA molecules, but **DNA-PKcs** did not inhibit ligation. Interestingly, **DNA-PKcs** stimulated intermolecular ligation by **DNA ligase IV-XRCC4**. In the presence of DNA-PK, the majority of the joining events catalyzed by **DNA ligase IV-XRCC4** were intermolecular because Ku inhibited intramolecular ligation, but **DNA-PKcs** still stimulated intramolecular ligation. We suggest that **DNA-PKcs**-containing complexes formed at DNA ends enhance the association of DNA ends via protein-protein interactions, thereby stimulating intermolecular ligation.
ACCESSION NUMBER: 2000458572 MEDLINE
DOCUMENT NUMBER: 20408964 PubMed ID: 10854421
TITLE: Interactions of the **DNA ligase IV-XRCC4** complex with DNA ends and the DNA-dependent protein kinase.
AUTHOR: Chen L; Trujillo K; Sung P; Tomkinson A E
CORPORATE SOURCE: Department of Molecular Medicine, Institute of Biotechnology, University of Texas Health Science Center, San Antonio, Texas 78245, USA.
CONTRACT NUMBER: ES07061 (NIEHS)
GM47251 (NIGMS)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 Aug 25) 275 (34)

26196-205.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009
ENTRY DATE: Entered STN: 20001005
Last Updated on STN: 20001005
Entered Medline: 20000925

L7 ANSWER 7 OF 28 MEDLINE
TI Defective embryonic neurogenesis in Ku-deficient but not DNA-dependent protein kinase catalytic subunit-deficient mice.
AB Mammalian nonhomologous DNA end joining employs Ku70, Ku80, DNA-dependent protein kinase catalytic subunit (**DNA-PKcs**), **XRCC4**, and **DNA ligase IV** (Lig4). Herein, we show that Ku70 and Ku80 deficiency but not **DNA-PKcs** deficiency results in dramatically increased death of developing embryonic neurons in mice. The Ku-deficient phenotype is qualitatively similar to, but less severe than, that associated with **XRCC4** and Lig4 deficiency. The lack of a neuronal death phenotype in **DNA-PKcs**-deficient embryos and the milder phenotype of Ku-deficient versus **XRCC4**- or Lig4-deficient embryos correlate with relative leakiness of residual end joining in these mutant backgrounds as assayed by a V(D)J recombination end joining assay. We conclude that normal development of the nervous system depends on the four evolutionarily conserved nonhomologous DNA end joining factors.
ACCESSION NUMBER: 2000183946 MEDLINE
DOCUMENT NUMBER: 20183946 PubMed ID: 10716994
TITLE: Defective embryonic neurogenesis in Ku-deficient but not DNA-dependent protein kinase catalytic subunit-deficient mice.
AUTHOR: Gu Y; Sekiguchi J; Gao Y; Dikkes P; Frank K; Ferguson D; Hasty P; Chun J; Alt F W
CORPORATE SOURCE: Howard Hughes Medical Institute, The Children's Hospital, and Center for Blood Research, and Department of Genetics, Harvard Medical School, Boston, MA 02115, USA.
CONTRACT NUMBER: A.I.01428 (NINDS)
A.I.35714
R01NS36949
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2000 Mar 14) 97 (6) 2668-73.
Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000505
Last Updated on STN: 20000505
Entered Medline: 20000425

L7 ANSWER 8 OF 28 MEDLINE
TI A new gene involved in DNA double-strand break repair and V(D)J recombination is located on human chromosome 10p.
AB V(D)J recombination, accountable for the diversity of T cell receptor- and immunoglobulin-encoding genes, is initiated by a lymphoid-specific DNA double-strand break. The general DNA repair machinery is responsible for the resolution of this break. Any defect in one of the known components of the DNA repair/V(D)J recombination machinery (Ku70, Ku80, **DNA-**

PKcs, **XRCC4** and **DNA ligase**
IV) leads to abortion of the V(D)J rearrangement process, early block in both T and B cell maturation, and ultimately to severe combined immune deficiency (SCID) in several animal models. A human SCID condition is also characterized by an absence of mature T and B lymphocytes, and is associated with an increase in sensitivity to DNA-damaging agents (RS-SCID). None of the above-mentioned genes are defective in these patients, arguing for the likelihood of the existence of yet another unknown component of the V(D)J recombination/DNA repair apparatus. Athabascan-speaking (SCIDA) Navajo and Apache Native Americans have a very high incidence of T(-)B(-)SCID. The SCIDA locus is highly linked with markers on chromosome 10p, although the exact molecular defect has not been recognized in these patients. We show here that cells with the SCIDA defect are impaired in the DNA repair phase of V(D)J recombination similarly to RS-SCID, precisely an absence of V(D)J coding joint formation. Moreover, genotyping analysis in several RS-SCID families corroborates a linkage of the RS-SCID locus to the SCIDA region on chromosome 10p. These results demonstrate the presence of a new essential DNA repair/V(D)J recombination gene in this region, the mutation of which causes RS-SCID in humans.

ACCESSION NUMBER: 2000164296 MEDLINE
DOCUMENT NUMBER: 20164296 PubMed ID: 10699181
TITLE: A new gene involved in DNA double-strand break repair and V(D)J recombination is located on human chromosome 10p.
AUTHOR: Moshous D; Li L; Chasseval R; Philippe N; Jabado N; Cowan M
J; Fischer A; de Villartay J P
CORPORATE SOURCE: Developpement Normal et Pathologique du Systeme Immunitaire, INSERM U429, Hopital Necker Enfants Malades, 149 rue de Sevres, 75015 Paris, France.
CONTRACT NUMBER: RO1 AI28339 (NIAID)
SOURCE: HUMAN MOLECULAR GENETICS, (2000 Mar 1) 9 (4) 583-8. Journal code: BRC; 9208958. ISSN: 0964-6906.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000427
Last Updated on STN: 20000427
Entered Medline: 20000414

L7 ANSWER 9 OF 28 MEDLINE
TI The catalytic subunit DNA-dependent protein kinase (**DNA-PKcs**) facilitates recovery from radiation-induced inhibition of DNA replication.
AB Exposure of cells to ionizing radiation inhibits DNA replication in a dose-dependent manner. The dose response is biphasic and the initial steep component reflects inhibition of replicon initiation thought to be mediated by activation of the S-phase checkpoint. In mammalian cells, inhibition of replicon initiation requires the ataxia telagiectasia mutated (ATM) gene, a member of the phosphatidyl inositol kinase-like (PIKL) family of protein kinases. We studied the effect on replicon initiation of another member of the PI-3 family of protein kinases, the catalytic subunit of DNA-dependent protein kinase (**DNA-PKcs**) by measuring either total DNA synthesis, or size distribution of nascent DNA using alkaline sucrose gradient centrifugation. Exposure of human cells proficient in **DNA-PKcs** (HeLa or M059-K) to 10 Gy inhibited replicon initiation in a time-dependent manner. Inhibition was at a maximum 1 h after irradiation and recovered at later times. Similar treatment of human cells deficient in **DNA-PKcs** (M059-J) inhibited replicon initiation to

a similar level and with similar kinetics; however, no evidence for recovery, or only limited recovery, was observed for up to 8 h after irradiation. In addition a defect was observed in the maturation of nascent DNA. Similarly, a Chinese hamster cell line deficient in **DNA-PKcs** (irs-20) showed little evidence for recovery of DNA replication inhibition up to 6 h after irradiation, whereas the parental CHO cells showed significant recovery and an irs-20 derivative expressing the human **DNA-PKcs** complete recovery within 4 h. Normal kinetics of recovery were observed in xrs-5 cells, deficient in Ku80; in 180BR cells, deficient in **DNA ligase IV**; as well as XR-1 cells, deficient in **XRCC4**, an accessory factor of **DNA ligase IV**. Since all these cell lines share the DNA double strand break rejoining defect of M059-J and irs20 cells, the lack of recovery of DNA replication in the latter cells may not be attributed entirely to the prolonged presence of unrepaired DNA dsb. We propose that **DNA-PKcs**, in addition to its functions in the rejoining of DNA dsb and in DNA replication, also operates in a pathway that in normal cells facilitates recovery of DNA replication after irradiation.

ACCESSION NUMBER: 2000133036 MEDLINE
DOCUMENT NUMBER: 20133036 PubMed ID: 10666461
TITLE: The catalytic subunit DNA-dependent protein kinase (**DNA-PKcs**) facilitates recovery from radiation-induced inhibition of DNA replication.
AUTHOR: Guan J; DiBiase S; Iliakis G
CORPORATE SOURCE: Department of Radiation Oncology of Kimmel Cancer Center, Thompson Building, Jefferson Medical College, Philadelphia, PA 19107, USA.
CONTRACT NUMBER: CA 42026 (NCI)
CA 56706 (NCI)
P30 CA56036=03 (NCI)
SOURCE: NUCLEIC ACIDS RESEARCH, (2000 Mar 1) 28 (5) 1183-92.
Journal code: O8L; 0411011. ISSN: 1362-4962.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000505
Last Updated on STN: 20010521
Entered Medline: 20000424

L7 ANSWER 10 OF 28 MEDLINE
TI Isolation of Ku70-binding proteins (KUBs).
AB DNA-dependent protein kinase (DNA-PK) plays a critical role in resealing DNA double-stand breaks by non-homologous end joining. Aside from DNA-PK, **XRCC4** and **DNA ligase IV**, other proteins which play a role(s) in this repair pathway remain unknown; DNA-PK contains a catalytic subunit (**DNA-PKcs**) and a DNA binding subunit (Ku70 and Ku80). We isolated Ku70-binding proteins (KUB1-KUB4) using yeast two-hybrid analyses. Sequence analyses revealed KUB1 to be apolipoprotein J (apoJ), also known as X-ray-inducible transcript 8 (XIP8), testosterone-repressed prostate message-2 (TRPM-2) and clusterin. KUB2 is Ku80. KUB3 and KUB4 are unknown, >10 kb trans-cripts. Interactions of apoJ/XIP8 or KUB3 with Ku70 were confirmed by co-immunoprecipitation analyses in MCF-7:WS8 breast cancer or IMR-90 normal lung fibroblast cells, respectively. The interaction of apoJ/XIP8 with Ku70 was confirmed by far-western analyses. Stable over-expression of full-length apoJ/XIP8 in MCF-7:WS8 caused decreased Ku70/Ku80 DNA end binding that was restored by apoJ/XIP8 monoclonal antibodies. The role of apoJ/XIP8 in ionizing radiation resistance/sensitivity is under investigation.

ACCESSION NUMBER: 1999238412 MEDLINE
DOCUMENT NUMBER: 99238412 PubMed ID: 10219089
TITLE: [redacted]lation of Ku70-binding proteins [redacted]KUBs).
AUTHOR: Yang C R; Yeh S; Leskov K; Odegaard E; Hsu H L; Chang C; Kinsella T J; Chen D J; Boothman D A
CORPORATE SOURCE: Department of Radiation Oncology and Department of Pharmacology and the Ireland Cancer Center, Laboratory of Molecular Stress Responses, Case Western Reserve University, BRB-326 East, 10900 Euclid Avenue, Cleveland, OH
44106-4942, USA.
CONTRACT NUMBER: CA-50595 (NCI)
CA-ES78530 (NCI)
CA50519 (NCI)
SOURCE: NUCLEIC ACIDS RESEARCH, (1999 May 15) 27 (10) 2165-74.
Journal code: O8L; 0411011. ISSN: 0305-1048.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990712
Last Updated on STN: 19990712
Entered Medline: 19990624